

Acute Cardiovascular Effects of Vaping Compared to Cigarette Smoking in Young Adults

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ABSTRACT

Background: First introduced as the safer alternative to smoking, vaping has become a popular activity among young adults. However, little is known about its potential health effects. This pilot project examined the acute cardiovascular effects of nicotine vapes/e cigarettes (EC) compared to tobacco cigarettes (TC) in young adults to determine if vaping is more detrimental to cardiovascular health than traditional cigarettes.

Methods: 16 healthy participants (7 M, 9 F; 20.2 ± 1.9 years) were recruited to participate in the study. Anthropometric measures were determined upon entry into the study. In addition, circulatory measures (heart rate [HR], blood pressure [BP] and heart rate variability [HRV]) were measured prior to and 10-min following vaping or cigarette smoking and in response to an orthostatic challenge.

Results: Resting circulatory and HRV measures were not different between chronic EC-users and TC-smokers. Both vaping and cigarette smoking-induced a significant increase in cardiovascular measures (HR and BP) but not HRV measures. Both groups responded similarly to the orthostatic challenge prior to and following vaping/smoking.

Conclusion: These results indicate that, from a cardiovascular perspective, vaping induces similar acute effects as cigarette smoking and that young adults should be counselled about these adverse effects accordingly.



KEYWORDS

Cigarettes, Vapes, Nicotine, Cardiovascular system, Adolescents

1 | INTRODUCTION

A substantial amount of literature indicates that tobacco cigarette (TC) smoking is related to a plethora of cardiovascular diseases (1); causing approximately 30% of car-

diovascular disease related deaths. (2) Smoking TC has been noted to increase an individual's chances of developing atherosclerotic diseases such as angina, acute coronary syndrome, stroke and sudden death. (3) Tobacco cigarettes contain over 7000 chemical compo-

nents, including nicotine (with concentrations of 1.99 ± 0.20 mg/cigarette), tar, carbon monoxide, acrolein and pro-oxidants. (4) Nicotine has been shown to be a predominant factor of accelerated atherogenesis and cardiovascular disease. (5)

Electronic cigarettes (e-cigarettes, EC) were introduced in 2006 as a more health-conscious alternative to cigarette smoking. (6) EC are electronic heating devices which create an aerosolized mixture of liquid containing stimulants (e.g., nicotine, marijuana), flavoring and solvents for heating that can be inhaled. (7) Nicotine concentrations in EC can range from 1.6 - 19 mg per cartridge. (8) The adverse effects of chronic EC use include bronchitis, emphysema, respiratory tract irritation and cardiovascular disease. (2)

It has been well established that the acute effects of smoking TC can change heart rate variability (HRV) parameters, leading to an increase in the low-frequency to high frequency (LF/HF) ratio. (9) This increase is attributable primarily to the effects of nicotine. (10) Individuals who smoke cigarettes regularly often experience a reduction in HRV, indicating an increased sympathetic nervous system (SNS) activity, increased heart rate (HR) and blood pressure (BP). (11) Similarly, vaping EC that contain nicotine has also been found to induce an increase in SNS activity. (10, 12)

While vaping is gaining popularity (4), little research has been done to determine its possible cardiovascular side effects (2) and minimal research has compared the effects of smoking tobacco cigarettes (TC) with vaping EC on HRV. (10) HRV is a reliable, non-invasive tool for determining autonomic nervous system control of the heart. In a study of 100 smokers (42 TC smokers and 58 chronic EC users), Arastoo et al (10) found that baseline cardiovascular and HRV measures were similar between TC-smokers and EC-users and that HR, systolic blood pressure [SBP], diastolic blood pressure [DBP], and mean arterial pressure [MAP] significantly increased following acute exposure to TC and EC. However, HRV measures were not altered. Interestingly, the increase in blood pressure [BP] was significantly greater in the TC smokers, which the authors believe may have been due to the greater number of chemicals contained in TC. So far,

other studies that have compared the acute autonomic effects of smoking TC with EC-use have examined HR, SBP and DBP as surrogate measures. (12) HR has been consistently shown to increase, whereas contradictory results have been reported for SBP and/or DBP. Similar to the work of Arastoo et al (10) a few studies have demonstrated that the cardiovascular autonomic effects of smoking TC are greater than vaping EC.

Moreover, no studies have examined the dynamic influence of a sympathetic maneuver (such as an orthostatic challenge) on HRV in TC-smokers and EC-users. One study with TC smokers examined the dynamic influence of parasympathetic (PNS) (controlled breathing) and sympathetic (SNS) maneuvers (hand-grip exercise) on HRV (13). Barutcu et al (13) found that parasympathetic (vagal) modulation was blunted in smokers during a controlled breathing exercise.

Therefore, the purpose of this study is to examine the acute (short-term) cardiovascular effects of cigarette smoking (TC) and e-cigarettes (EC). This research is important as EC are becoming increasingly popular among cigarette smokers and those who have no previous history of smoking, yet, there are gaps in the literature examining the acute health consequences of regular EC use. (2) This project examined the acute cardiovascular effects of smoking TC in young adults in comparison to those who vape EC as well as their response to a postural challenge. Based upon our review of the literature, we hypothesized that there would be no difference in resting circulatory and HRV parameters between TC smokers and EC-users that heart rate and blood pressure would significantly increase following cigarette-smoking and vaping without measurable changes in HRV and that both groups would respond similarly to an orthostatic challenge. Since it was reported that the perturbation in circulatory measures tends to be greater for individuals who use TC vs EC (10), we predicted that individuals in the TC group would have a higher HR, blood pressure, and increased sympathetic tone compared to individuals who vape EC.

2 | METHODS

This study used a pre-test, post-test cross-sectional design to assess the cardiovascular effects of smoking versus vaping. A flow chart representing the experimental design and outcome is depicted in **Figure 1**. The study was conducted between the months of January and March of 2020 and was approved by Trent University's Research Ethics Board (File 26045) in accordance with the Declaration of Helsinki.

2.1 | Participants

Participants were recruited through announcements made in class and posters placed at a post-secondary institution in Ontario, Canada. Interested participants contacted the research team. Exclusion criterion included the presence of known disease (e.g., cardiorespiratory, neurological, gastrointestinal, metabolic and psychiatric). Nineteen participants who either smoked traditional TC or used nicotine EC volunteered to participate in the study. Three participants were excluded from the study (one participant had a medical condition and two participants could not return for testing due to the COVID-19 pandemic lockdown). Participants ranged between the ages of 18 through 25 years and were assigned to their respective groups (TC vs EC) based upon their smoking history (a minimum of a 1-year smoking or vaping requirement for participation). The goal was to have exclusive smokers/vapers in the study, however, 4 smokers and 3 vapers disclosed that they occasionally vaped/smoked, respectively.

2.2 | Procedures

Participants visited the laboratory on two occasions. On the first visit, the experimental procedure was explained and written, informed consent was obtained. The participants then completed a questionnaire that was used to gather demographics and smoking/vaping history.

Anthropometric measures were taken including body mass, height, and skinfold measures. Body mass (kilogram [kg]) and height (metres [m]) were obtained us-

ing a Health-O-Meter scale (Health-O-Meter Corporation, Bedford Heights, Ohio). Body mass index (BMI) was determined by dividing the body mass (kg) by the participant's height (m^2). Body density was determined through skinfold measures, taken using a Harpenden skinfold caliper (FitSystems, Inc., Calgary, AB), from seven sites of the body (abdomen, biceps, thigh, iliac crest, midaxillary, chest, and subscapula) according to the Jackson-Pollock formulas for men and women. (14) Percent body fat was calculated from body density using the Siri body density conversion formula.

One week later, on the same day and time of the week, participants returned to the laboratory to have cardiovascular measures recorded. HR, SBP and DBP were determined in the supine and standing positions (prior to and following smoking/vaping) using an automated, non-invasive BP monitor (BpTRU) (Model BPM-300, VSM Medtech Ltd., Coquitlam, BC). Mean arterial pressure (MAP) and rate pressure product (RPP) were also determined. MAP, an indicator of the average blood pressure during one cardiac cycle was calculated from: $MAP = 1/3 (SBP-DBP) + DBP$. Rate pressure product (RPP), a measure of the workload of the heart, was determined from the product of heart rate and systolic blood pressure. Beat-by-beat R-R intervals were recorded continuously using a Polar Sport HR chest strap which transmitted the data to a wristwatch (Polar Vantage V2, Polar Sport, Montreal, QC).

For the supine condition, participants rested comfortably, in the supine position, on an examination table located in a quiet, light attenuated room. Participants refrained from speaking and moving for 10 min to allow for the recording of the cardiovascular measures. Cardiovascular measures were then recorded with the participant standing in an upright position for 10 minutes. Participants remained in the freestanding position without support for an adaptation period of 3 minutes followed by 10 minutes (or 512 heart cycles) of beat-by-beat data recording. Participants were reminded to relax, remain as still as possible, avoid talking and asked to refrain from leaning backwards on the examination table that was located behind them. Once the initial measures were obtained in the supine and standing po-

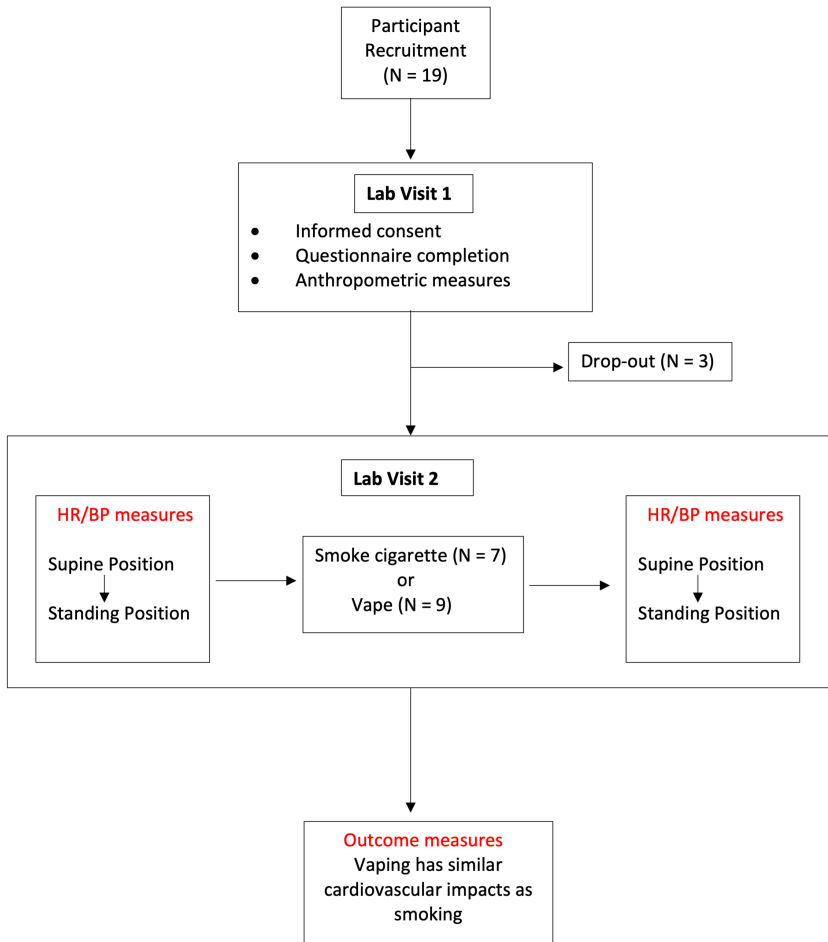


FIGURE 1 Study experimental design.

sitions, the participants were escorted to a designated smoking/vaping area. Participants were given a rest period of at least 3 minutes after reaching the smoking/vaping area so that their heart rate could return to baseline before smoking or vaping. The participants then either vaped e-cigarettes with 5.0% nicotine or smoked their normal cigarettes in the seated position for 2 and 5 minutes, respectively. Following this, participants returned to the laboratory to have their cardiovascular measures recorded again in the supine and standing positions. Polar Sport HR monitor recorded the R-R intervals. We determined HRV using Kubios (Kubios Oy, Kuopio, Finland).

2.3 | Statistical Analysis

Data were statistically analyzed using the computer program IBM SPSS Statistics for Macintosh, Version 27.0 (IBM Corp, Armonk, NY). Data are presented as means (\pm SD). Descriptive measures were assessed using an independent t-test or Chi-square test, whereas the hemodynamic and HRV variables of the TC smokers were compared to that of the EC users using a repeated measures ANOVA (within-group measure = time; between-group measure = TC vs EC). A p-value of 0.05 or less was considered significant.

3 | RESULTS

3.1 | Baseline characteristics of participants were similar

No significant differences were observed in baseline characteristics of the seven chronic TC smokers and the nine chronic EC-users, with the exception of years smoking or vaping (Table 1). Those individuals who smoked TC did so for a significantly ($p = 0.017$) longer period of time (4.7 years) compared to individuals who vaped EC (1.7 years). More than half (56%) of the participants were women, and they had a significantly ($p = 0.041$) higher BMI, compared to men, with an average BMI of 28.7 (9.3) versus 21.1 (2.33) kg/m². This pattern was more notable in the vaping (EC) group, albeit non-significant ($p = 0.066$), whereby BMI was 32.6 (10.5) for women compared to 20.9 (2.99) for men. In the group of TC smokers, BMI was 23.9 (5.4) for women versus 21.34 (1.7) for men.

3.2 | Cardiovascular responses were similar between cigarette smokers and vapers

The cardiovascular measures are presented in Table 2. There were no significant between-group (TC vs EC)

differences in the resting cardiovascular measures, response to smoking/vaping or to the orthostatic challenge.

Repeated measures ANOVA indicated that HR ($F(1,14) = 12.740$, $p = 0.003$), SBP ($F(1,14) = 16.980$, $p < 0.001$), DBP ($F(1,14) = 23.502$, $p < 0.001$), MAP ($F(1,14) = 29.797$, $p < 0.001$) and RPP ($F(1,14) = 30.028$, $p < 0.001$) significantly increased in response to smoking/vaping. Compared to the resting condition, HR ($F(1,14) = 382.164$, $p < 0.001$), SBP ($F(1,14) = 11.070$, $p = < 0.001$), (DBP $F(1,14) = 55.746$, $p = < 0.001$), MAP ($F(1,14) = 55.304$, $p = < 0.001$) and RPP ($F(1,14) = 133.320$, $p = < 0.001$) were significantly increased in response to standing prior to smoking/vaping. Likewise, HR ($F(1,14) = 111.164$, $p = < 0.001$), SBP ($F(1,14) = 25.748$, $p = < 0.001$), DBP ($F(1,14) = 136.310$, $p = < 0.001$), MAP ($F(1,14) = 115.010$, $p = < 0.001$) and RPP ($F(1,14) = 161.092$, $p = < 0.001$) significantly increased in response to standing after smoking/vaping.

3.3 | HRV measures were significantly influenced by postural change

The HRV measures are presented in Table 3. No between-group (TC vs EC) differences were observed for the LF power, HF power, total power and the SNS in-

Variable	TC (n = 7)	EC (n = 9)	p value	ALL (n = 16)
Age (yrs)	21.1 (2.12)	19.4 (1.4)	0.075	20.2 (1.9)
Male sex (%)	3 (43)	4 (44)	0.949	7 (44)
Height (m)	1.7 (1.3)	1.8 (1.4)	0.528	1.7 (1.4)
Weight (kg)	66.5 (11.2)	82.3 (22.0)	0.087	75.4 (19.4)
BMI (kg/m ²)	22.8 (4.2)	27.4 (9.8)	0.235	25.4 (8.0)
Percent fat (%)	15.6 (11.8)	20.5 (10.8)	0.395	18.4 (11.2)
HR (bpm)	79.6 (9.8)	72.9 (13.5)	0.288	75.9 (12.1)
Systolic BP (mmHg)	108.8 (8.4)	109.7 (5.7)	0.794	109.3 (6.8)
Diastolic BP (mmHg)	72.2 (6.0)	71.9 (6.0)	0.910	72.0 (5.8)
Smoking/vaping (yrs)	4.7 (2.8)	1.7 (1.7)	0.017*	3.00 (2.66)
Smokes/vapes (/day)	3.7 (1.7)	4.0 (1.5)	0.727	3.88 (1.54)

Legend: Values are means \pm standard deviation (SD). BMI = body mass index, BP = blood pressure, HR = heart rate. * = $p < 0.05$

TABLE 1 Descriptive characteristics of participants at entry into the study.

Variable	Condition	Before smoking/vaping			After smoking/vaping		
		TC (n = 7)	EC (n = 9)	All (n = 16)	TC (n = 7)	EC (n = 9)	All (n = 16)
HR (bpm)	Rest	79.6 (9.8)	72.9 (13.5)	75.9 (12.1)	87.0 (13.1)	76.1 (14.6)	80.8 (14.6)**
	Stand	97.3 (14.8)	94.1 (13.8)	95.5 (13.8)*	101.4 (13.4)	94.8 (12.8)	97.7 (13.1)***
SBP (mmHg)	Rest	108.8 (8.4)	109.7 (5.7)	109.3 (6.8)	117.9 (8.0)	118.2 (8.6)	118.1 (8.1)**
	Stand	111.3 (7.8)	112.0 (5.7)	111.7 (6.5)*	114.4 (6.19)	119.1 (7.5)	117.0 (7.1)***
DBP (mmHg)	Rest	72.2 (6.0)	71.9 (6.0)	72.0 (5.8)	78.1 (6.9)	76.2 (7.4)	77.0 (7.0)**
	Stand	81.3 (4.3)	79.4 (6.5)	80.3 (5.6)*	83.5 (5.8)	84.5 (6.0)	84.0 (5.7)***
MAP (mmHg)	Rest	84.4 (6.0)	84.5 (5.5)	84.5 (5.6)	91.3 (6.0)	90.2 (7.1)	90.7 (6.5)**
	Stand	91.3 (5.1)	90.3 (5.9)	90.7 (5.4)*	93.8 (5.0)	96.0 (5.6)	95.0 (5.3)***
RPP (x 10 ³) (bpm.mmHg ⁻¹)	Rest	8.6 (1.2)	8.0 (1.7)	8.3 (1.5)	10.2 (1.5)	9.0 (2.0)	9.5 (1.8)**
	Stand	10.8 (1.6)	10.6 (1.8)	10.7 (1.7)*	11.6 (1.6)	11.3 (1.8)	11.4 (1.7)***

Legend: Values are means \pm (SD). Abbreviations are as follows: DBP = diastolic blood pressure; EC = electronic cigarette user; HR = Heart rate; SBP = systolic blood pressure; MAP = mean arterial blood pressure; RPP = rate pressure product; TC = tobacco cigarette smoker. * = $p < 0.05$ ("all" stand condition vs "all" rest condition - before smoking/vaping); ** $p < 0.05$ = ("all" rest condition after smoking/vaping vs "all" rest condition before smoking/vaping); *** $p < 0.05$ ("all" stand condition after smoking/vaping vs "all" rest condition before smoking/vaping).

TABLE 2 Circulatory responses of the participants.

indicator (LF/HF) in the resting condition and in response to smoking/vaping. There was a significant between-group effect for the PNS indicator (HF/total power) ($F(1,14) = 7.735$, $p < 0.015$) with the orthostatic challenge. Standing upright had a stronger PNS effect in the vaping group (both prior to and following vaping).

Repeated measures ANOVA indicated a significant main effect of standing on several autonomic indices. Compared to resting, both the HF ($F(1,14) = 5.936$, $p < 0.029$) and PNS (HF/total) ($F(1,14) = 25.921$, $p < 0.001$) indicator were significantly reduced with standing prior to smoking/vaping. Similarly, the HF ($F(1,14) = 8.047$, $p < 0.013$) and PNS (HF/total) ($F(1,14) = 35.307$, $p < 0.001$) indicator were significantly reduced with standing after smoking/vaping. In contrast, the SNS indicator was significantly increased in response to standing both before ($F(1,14) = 43.1$, $p < 0.001$) and after ($F(1,14) = 33.622$, $p < 0.001$) smoking/vaping.

4 | DISCUSSION

The major findings of this study are that resting hemodynamics and HRV measures are similar between chronic

TC smokers and EC vapers; HR and BP significantly increase in response to smoking and vaping, but HRV measures remain the same; and that the autonomic response to orthostatic challenge is not altered by smoking or vaping. These results support our hypotheses. However, our prediction that the circulatory responses of the TC-smokers would be greater was not observed.

To better comprehend the acute cardiovascular effects of smoking, HRV is often assessed. (11) HRV can be measured in both the time- and frequency-domain. Time domain analysis quantifies the R-R interval between two neighboring heartbeats over a set period of time. Frequency domain analysis is commonly used for analysis of brief recordings of heart rate (HR). (15) In a frequency-domain analysis, the variability in HR is put through a mathematical model (Fast-Fourier transformation) to provide indicators of autonomic function. High frequency (HF) power values are associated with parasympathetic activity, and low frequency power (LF) is correlated to both sympathetic and parasympathetic nervous system activity. (11) Parasympathetic nervous system (PNS) modulation can be inferred from the parasympathetic indicator (a ratio of the high frequency to total power), whereas sympathetic (SNS) modulation

Variable	Condition	Before smoking/vaping			After smoking/vaping		
		TC (n = 7)	EC (n = 9)	All (n = 16)	TC (n = 7)	EC (n = 9)	All (n = 16)
LF (ms ² /Hz)	Rest	824 (627)	2825 (5856)	1950 (4416)	403 (194)	2663 (5107)	1674 (3907)
	Stand	1466 (1681)	2740 (3364)	2182 (2755)	1658 (2388)	1979 (3348)	2182 (2755)
HF (ms ² /Hz)	Rest	617 (620)	1532 (1730)	1132 (1404)	394 (450)	2603 (4814)	1637 (3704)
	Stand	164 (154)	690 (1343)	460 (1022)*	162 (153)	496 (953)	350 (723)***
Total Power (ms ² /Hz)	Rest	2654 (1325)	5067 (7002)	4012 (5327)	1868 (836)	6452 (11506)	4447 (8741)
	Stand	2724 (2429)	5068 (6796)	4043 (5332)	2621 (2674)	3767 (6432)	3265 (5027)
PNS Indicator (ms ² /Hz)	Rest	0.20 (0.14)	0.34 (0.16)	0.28 (0.16)	0.19 (0.14)	0.40 (0.24)	0.31 (0.22)
	Stand	0.06 (0.02)	0.12 (0.05)†	0.08 (0.04)*	0.06 (0.04)	0.12 (0.07)††	0.09 (0.06)***
SNS Indicator (ms ² /Hz)	Rest	3.39 (4.67)	1.64 (1.50)	2.40 (3.27)	1.78 (1.45)	1.24 (0.75)	1.47 (1.10)
	Stand	8.60 (4.54)	6.21 (2.91)	7.26 (3.77)*	8.70 (3.64)	5.76 (2.43)	7.05 (3.27)***

Legend: EC = Electronic cigarettes; HF = High frequency, LF = low frequency, PNS = parasympathetic nervous system, SNS = sympathetic nervous system, TC = tobacco cigarettes, * = $p < 0.05$ ("all" stand condition vs "all" rest condition - before smoking/vaping), *** = $p < 0.05$ ("all" stand condition after smoking/vaping vs "all" rest condition before smoking/vaping); † = $p < 0.05$ between group effect (TC vs EC before smoking/vaping, rest to stand transition); †† = $p < 0.05$ between group effect (TC vs EC after smoking/vaping, rest to stand transition).

TABLE 3 HRV responses of the participants.

can be inferred from the sympathetic indicator (a ratio of low frequency to high frequency power).

Our findings of similar resting circulatory and HRV measures between chronic TC-smokers and EC-users is supported by the work of Arastoo et al (10) who examined baseline, resting hemodynamics and HRV measures in 100 participants (42 chronic TC-smokers and 58 chronic EC-users). These authors found no difference between the groups on circulatory and HRV measures. They attributed these results to a consistent level of cardiac sympathetic activity within the two groups.

Secondly, this study demonstrated that circulatory parameters including HR, SBP and DBP as well as derivatives of these variables (MAP and RPP) were all significantly increased 10 min following the smoking as well as the vaping sessions. However, HRV measures were unchanged. Several studies compared the acute circulatory/hemodynamic responses in TC-smokers with EC-users. (8, 10, 16-20) One study also examined the acute effects of smoking/vaping on HRV. (10) Studies which have kept nicotine exposure equivalent between the groups, revealed that HR and/or BP are significantly increased to a greater extent in TC-smokers compared to EC-users. (8, 10, 16, 20) This augmented effect of TC-

smoking has been attributed to the greater number of chemicals (e.g., tar) in TC smoke. (10) These results indicate that TC-smoking may have a greater detrimental effect on the circulatory system and support the use of EC as a safer alternative to TC-smoking or as a smoking cessation strategy.

The findings of our study, however, agree with the findings of Franzen et al (17) and Vlachopoulos et al (19) whereby the increases in HR, SBP and DBP were comparable between the two groups. In these studies, the nicotine delivery may have been actually greater in the EC-use group leading to the equivalent increases in circulatory measures. Franzen et al (17) performed a randomized cross-over study with 15 active smokers. The physiological responses to TC smoking or EC-use (either with nicotine [24 mg/mL] or without nicotine) were examined in random order. HR, SBP, and DBP increased similarly in the nicotine conditions and but not in the EC condition without nicotine. The intensity of vaping was not standardized according to the depth of each breath during a puff. Thus, the amount of nicotine exposure could not be controlled. Vlachopoulos et al (19) also performed a randomized cross-over study with 24 TC smokers. Participants smoked either a TC or vaped

an EC without knowing the nicotine content. HR, SBP and DBP were monitored for 5-min as well as 30-min. Increases in HR, SBP and DBP were similar between smoking and vaping conditions at the 30-minute mark. Plasma nicotine levels were not assessed.

Both the tobacco cigarettes as well as the e-cigarettes used in this study contained nicotine. Nicotine stimulates the release of catecholamines and neurotransmitters and can cause tachycardia, and hypertension. (21) Nicotine also exerts pharmacologic effects which can enhance cardiac sympathetic activity. (5) Our finding of a negligible change in HRV dynamics in response to smoking/vaping is supported by the findings of Arastoo and colleagues. (10) These authors propose that the lack of change in HRV measures may be due to chronic tolerance of nicotine (by chronic smokers/vapers) and desensitization of central nicotinic receptors. Although sympathetic activity (as reflected by the SNS indicator) was not increased 10 minutes post-smoking/vaping, it is also possible that this parameter was increased during the smoking/vaping session and that this led to the increase observed in circulatory parameters. Moreover, withdrawal of PNS activity may also have contributed to the observed effects.

Finally, a unique aspect of this study was examining the circulatory and HRV response of smokers and vapers to an orthostatic challenge. Both groups responded similarly to the challenge, both before and after smoking/vaping. Postural change from the supine position to the standing position will induce a drop in BP due to venous pooling. (22) This drop in BP is detected by the baroreceptors which, in turn, will stimulate an increase in HR (via SNS activation and PNS withdrawal) and consequently will induce an increase in BP. This response was observed in this study and was not altered by smoking and vaping. It is also noteworthy that the orthostatic challenge placed a much greater physiological demand on the body than smoking/vaping.

4.1 | Limitations

This was a pilot study with a cross-sectional design and a small sample size ($n = 16$; TC = 7 and EC = 9). A

cross-over study could have increased the sample size and reduce the variability between the two groups. Secondly, a non-smoking comparison group was not included in the current study; a comparison group would have been useful to determine if the resting baseline circulatory HRV measures were increased due to an elevation in sympathetic activity. One study which examined resting HRV in healthy adults (aged 28 ± 8 years) indicated lower resting measures for HR (64 ± 10 bpm), SBP (108 ± 12 mmHg), DBP (64 ± 8 mmHg) and values for LF power ($734 [247-2389]$ ms²) compared to the data obtained in our study. (23) It is also important to note that there are multiple factors which can influence changes in HRV such as; lifestyle, environmental, physiological, and neuropsychological factors. (24) These factors could have influenced our results. Also, we were unable to regulate the concentration of nicotine within the participant's cigarettes and vapes. In Canada, tobacco cigarettes contain between 8.0 – 18.3 mg (13.5 ± 0.49 mg) of tobacco which translates to 1.0 – 2.4 % ($1.80 \pm 0.06\%$) tobacco/cigarette. (25) The majority of EC users in this study vaped 5.0% nicotine (59 mg/ml) however two individuals used 3.0 % (39 mg/ml). (26) Ideally, blood samples to check for plasma nicotine concentrations following the smoke/vape session would have been obtained.

5 | CONCLUSIONS

The current study demonstrates that vaping has the same acute cardiovascular effects as smoking cigarettes in a small group of young adults. Thus, the use of e-cigarettes (vaping) containing nicotine may not be the safer alternative to cigarette smoking that is advertised. This discrepancy is significant as many individuals begin vaping because they believe it to be better for their health than cigarette smoking. With the increase in teens and young adults beginning to vape (27) more research on the cardiovascular response is needed to determine the relative safety and health risks associated with this behavior. It is important to educate the public regarding these risks, to make informed decisions re-

garding their health.

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REFERENCES

- Ockene IS, Miller NH. Cigarette smoking, cardiovascular disease, and stroke. *Circulation*. 1997;96(9):3243-7. Available from <https://doi.org/10.1161/01.CIR.96.9.3243>
- Qasim H, Karim ZA, Rivera JO, Khasawneh FT, Alshbool FZ. Impact of electronic cigarettes on the cardiovascular system. *Journal of the American Heart Association*. 2017;6(9). Available from <https://doi.org/10.1161/JAHA.117.006353>
- Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: An update. *Journal of the American College of Cardiology*. 2004;43(10):1731-7. Available from <https://doi.org/10.1016/j.jacc.2003.12.047>
- George J, Hussain M, Vadiveloo T, Ireland S, Hopkinson P, Struthers AD, et al. Cardiovascular effects of switching from tobacco cigarettes to electronic cigarettes. *Journal of the American College of Cardiology*. 2019;74(25):3112-20. Available from <https://doi.org/10.1016/j.jacc.2019.09.067>
- Benowitz NL, Burbank AD. Cardiovascular toxicity of nicotine: Implications for electronic cigarette use. *Trends in Cardiovascular Medicine*. 2016;26(6):515-23. Available from <https://dx.doi.org/10.1016/j.tcm.2016.03.001>
- Bhatnagar A. Are electronic cigarette users at increased risk for cardiovascular disease? *JAMA Cardiol*. 2017;2(3):237-8. Available from <https://dx.doi.org/10.1001/jamacardio.2016.5550>
- Wu JC, Rhee J-W, Sallam K. Electronic cigarettes. Where there is smoke there is disease. *Journal of the American College of Cardiology*. 2019;74(25):3121-3. Available from <https://www.jacc.org/doi/full/10.1016/j.jacc.2019.10.029>
- Biondi-Zoccai G, Sciarretta S, Bullen C, Nocella C, Violi F, Lofredo L, et al. Acute effects of heat-not-burn, electronic vaping, and traditional tobacco combustion cigarettes: The Sapienza University of Rome- Vascular Assessment of Proatherosclerotic Effects of Smoking (SUR- VAPES) 2 Randomized Trial. *Journal of the American Heart Association*. 2019;8:e010455. Available from <https://dx.doi.org/10.1161/JAHA.118.010455>
- Karakaya O, Barutcu I, Kaya D, Esen AM, Saglam M, Melek M, et al. Acute effects of cigarette smoking on heart rate variability. *Angiology*. 2007;58(5):620-4. Available from <https://doi.org/10.1177/0003319706294555>
- Arastoo S, Haptonstall KP, Choroomi Y, Moheimani R, Nguyen K, Tran E, et al. Acute and chronic sympathomimetic effects of e-cigarette and tobacco cigarette smoking: role of nicotine and non-nicotine constituents. *American Journal of Physiology Heart and Circulatory Physiology*. 2020;319(2). Available from <https://doi.org/10.1152/ajpheart.00192.2020>
- Lee CL, Chang WD. The effects of cigarette smoking on aerobic and anaerobic capacity and heart rate variability among female university students. *International Journal of Women's Health*. 2013;5:667-79. Available from <https://doi.org/10.2147/IJWH.S49220>
- Garcia PD, Gornbein JA, Middlekauff HR. Cardiovascular autonomic effects of electronic cigarette use: a systematic review. *Clinical Autonomic Research*. 2020;30:507-19. Available from <https://doi.org/10.1007/s10286-020-00683-4>
- Barutcu I, Esen AM, Kaya D, Muhsin T, Karakaya O, Melek M, et al. Cigarette smoking and heart rate variability: Dynamic influence of parasympathetic and sympathetic maneuvers. *A N E*. 2005;10(3):324-9. Available from <https://doi.org/10.1111/j.1542-474X.2005.00636.x>
- Acevedo EO, Starks MA. Exercise testing and prescription lab manual. 2nd Ed. Windsor, ON: Human Kinetics; 2011.
- Lewis MJ. Heart rate variability analysis: a tool to assess cardiac autonomic function. *Computers, Informatics, Nursing*. 2005;23(6):335-41. Available from <https://doi.org/10.1097/00024665-200511000-00011>
- Farsalinos KE, Tsiapras D, Kyrzopoulos S, Savvopoulou M, Voudris V. Acute effects of using an electronic nicotine-delivery device (electronic cigarette) on myocardial function: comparison with the effects of regular cigarettes. *BMC Cardiovascular Disorders*. 2014;14(78). Available from <http://www.biomedcentral.com/1471-2261/14/78>
- Franzen KF, Willig J, Talavera SC, Meusel M, Sayk F, Reppel M, et al. E-cigarettes and cigarettes worsen peripheral and central hemodynamics as well as arterial stiffness: A randomized, double-blinded pilot study. *Vascular Medicine*. 2018;23(5):419-25. Available from <https://doi.org/10.1177/1358863X18779694>
- Ikonomidis I, Vlastos D, Kourea K, Kostelli G, Varoudi M, Pavlidis G, et al. Electronic cigarette smoking increases arterial stiffness and oxidative stress to a lesser extent than a single conventional cigarette. An acute and chronic study. *Circulation*. 2018;137:303-6. Available from <https://doi.org/10.1161/CIRCULATIONAHA.117.029153>
- Vlachopoulos C, Ioakeimidis N, Abdelrasoul M, Terentes-Printzios D, Georgakopoulos C, Pietri P, et al. Electronic cigarette smoking increases aortic stiffness and blood pressure in young smokers. *Journal of the American College of Cardiology*. 2016;67(23):2802-3. Available from <https://doi.org/10.1016/j.jacc.2016.03.569>
- Vansickel AR, Cobb CO, Weaver MF, Eissenberg TE. A clinical laboratory model for evaluating the acute effects of electronic "cigarettes": Nicotine delivery profile and cardiovascular and sub-

- jective effects. *Cancer Epidemiology, Biomarkers Prevention*. 2010;19(8):1945-53. Available from <https://doi.org/10.1158/1055-9965.EPI-10-0288>
21. Bullen C. Impact of tobacco smoking and smoking cessation on cardiovascular risk and disease. *Expert Review of Cardiovascular Therapy*. 2008;6(6):883-95. Available from <https://doi.org/10.1586/14779072.6.6.883>.
22. Butler GC, Yamamoto Y, Xing C, Northery DR, Hughson RL. Heart rate variability and fractal dimension during orthostatic challenges. *Journal of Applied Physiology*. 1993;75(6): 2602-12. Available from <https://doi.org/10.1152/jappl.1993.75.6.2602>.
23. Keet, SW, Bukte CS, Garnier RP, Boer C, Bouwmann, RA. Short-term heart rate variability in healthy adults. *Anaesthesia*. 2013;68(7):775-7. Available from [doi:10.1111/anae.12317](https://doi.org/10.1111/anae.12317), [10.1111/anae.12317](https://doi.org/10.1111/anae.12317).
24. Fatisson J, Oswald V, Lalonde F. Influence diagram of physiological and environmental factors affecting heart rate variability: an extended literature overview. *Heart international*. 2016;11(1):heartint-5000232.
25. Kozłowski LT, Mehta NY. Filter ventilation and nicotine content of tobacco in cigarettes from Canada, the United Kingdom, and the United States. *Tobacco Control*. 1998;7:369-75. Available from <http://dx.doi.org/10.1136/tc.7.4.369>
26. Omaiye EE, McWhirter KJ, Luo W, Pankow JF, Talbot P. High-nicotine electronic cigarette products: Toxicity of JUUL fluids and aerosols correlates strongly with nicotine and some flavor chemical concentrations. *Chemical Research in Toxicology*. 2019;32(6):1058-69. Available from <https://doi.org/10.1021/acs.chemrestox.8b00381>
27. Jones K, Salzman GA. The vaping epidemic in adolescents. *Missouri Medicine*. 2020;117(1):56-8. Available from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7023954/>